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 NEWS  1          Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2  Dec 17  The CA Lexicon available in the CAPLUS and CA files
 NEWS  3  Feb 06  Engineering Information Encompass files have new names
 NEWS  4  Feb 16  TOXLINE no longer being updated
 NEWS  5  Apr 23  Search Derwent WPINDEX by chemical structure
 NEWS  6  Apr 23  PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
 NEWS  7  May 07  DGENE Reload
 NEWS  8  Jun 20  Published patent applications (A1) are now in USPATFULL
 NEWS  9  JUL 13  New SDI alert frequency now available in Derwent's
                  DWPI and DPCI
 NEWS 10  Aug 23  In-process records and more frequent updates now in
                  MEDLINE
 NEWS 11  Aug 23  PAGE IMAGES FOR 1947-1966 RECORDS IN CAPLUS AND CA
 NEWS 12  Aug 23  Adis Newsletters (ADISNEWS) now available on STN
 NEWS 13  Sep 17  IMSworld Pharmaceutical Company Directory name change
                  to PHARMASEARCH
 NEWS 14  Oct 09  Korean abstracts now included in Derwent World Patents
                  Index
 NEWS 15  Oct 09  Number of Derwent World Patents Index updates increased
 NEWS 16  Oct 15  Calculated properties now in the REGISTRY/ZREGISTRY File
 NEWS 17  Oct 22  Over 1 million reactions added to CASREACT
 NEWS 18  Oct 22  DGENE GETSIM has been improved
 NEWS 19  Oct 29  AAASD no longer available
 NEWS 20  Nov 19  New Search Capabilities  USPATFULL and USPAT2
 NEWS 21  Nov 19  TOXCENTER(SM) - new toxicology file now available on STN
 NEWS 22  Nov 29  COPPERLIT now available on STN
 NEWS 23  Nov 29  DWPI revisions to NTIS and US Provisional Numbers
 NEWS 24  Nov 30  Files VETU and VETB to have open access
 NEWS 25  Dec 10  WPINDEX/WPIDS/WPIX New and Revised Manual Codes for 2002
 NEWS 26  Dec 10  DGENE BLAST Homology Search
 NEWS 27  Dec 17  WELDASEARCH now available on STN
 NEWS 28  Dec 17  STANDARDS now available on STN
 NEWS 29  Dec 17  New fields for DPCI
 NEWS 30  Dec 19  CAS Roles modified
 NEWS 31  Dec 19  1907-1946 data and page images added to CA and CAplus

 NEWS EXPRESS  August 15 CURRENT WINDOWS VERSION IS V6.0c,
               CURRENT MACINTOSH VERSION IS V6.0 (ENG) AND V6.0J (JP),
               AND CURRENT DISCOVER FILE IS DATED 07 AUGUST 2001
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS INTER    General Internet Information
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 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)

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specific topic.

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FILE 'HOME' ENTERED AT 13:29:27 ON 07 JAN 2002

=> file medline caplus embase biosis
COST IN U.S. DOLLARS                                  SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.15       0.15

FILE 'MEDLINE' ENTERED AT 13:29:38 ON 07 JAN 2002

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FILE 'BIOSIS' ENTERED AT 13:29:38 ON 07 JAN 2002
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=> s (((IL12 (1N) receptor) or (IL-12 (1N) receptor)) (2N) beta? ) 10N monoclonal
MISSING OPERATOR ) 10N
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s ((((IL12 (1N) receptor) or (IL-12 (1N) receptor)) (2N) beta? ) 10N monoclonal
MISSING OPERATOR ) 10N
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s ((((IL12 (1N) receptor) or (IL-12 (1N) receptor)) (2N) beta? ) (10N) monoclonal
UNMATCHED LEFT PARENTHESIS '(('
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s (((IL12 (1N) receptor) or (IL-12 (1N) receptor)) (2N) beta? ) (10N) monoclonal
L1            1 (((IL12 (1N) RECEPTOR) OR (IL-12 (1N) RECEPTOR)) (2N) BETA? )
                (10N) MONOCLONAL

=> dis l1
```

```
L1   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2002 ACS
AN   2000:688272  CAPLUS
DN   133:280563
TI   Human antibodies that bind human IL-12 and methods for producing
IN   Salfeld, Jochen G.; Roguska, Michael; Paskind, Michael; Banerjee,
     Subhashis; Tracey, Daniel E.; White, Michael; Kaymakcalan, Zehra;
     Labkovsky, Boris; Sakorafas, Paul; Friedrich, Stuart; Myles, Angela;
     Veldman, Geertruida M.; Venturini, Amy; Warne, Nicholas W.; Widom, Angela;
     Elvin, John G.; Duncan, Alexander R.; Derbyshire, Elaine J.; Carmen, Sara;
     Smith, Stephen; Holtet, Thor Las; Du, Fou Sarah L.
PA   Basf A.-G., Germany; Genetics Institute Inc.; et al.
SO   PCT Int. Appl., 377 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.         KIND  DATE           APPLICATION NO.  DATE
     ---------------    ----  --------       ---------------  --------
PI   WO 2000056772      A1    20000928       WO 2000-US7946   20000324
         W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR,
            CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU,
            ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU,
            LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE,
            SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA,
            ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
            DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
            CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRAI US 1999-126603     P     19990325
RE.CNT  7
RE
(2) Carter, R; HYBRIDOMA 1997, V16(4), P363 CAPLUS
(3) Genentech Inc; WO 9404679 A 1994 CAPLUS
(4) Genetics Inst; WO 9524918 A 1995 CAPLUS
(5) Irving, R; IMMUNOTECHNOLOGY 1996, V2(2), P127 CAPLUS
(6) Pini, A; JOURNAL OF IMMUNOLOGICAL METHODS 1997, V206(1-2), P171 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> dis l1 kwic

L1   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2002 ACS


=> dis l1  1 kwic

L1   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2002 ACS


=> dis l1 ibib abs kwic

L1   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:        2000:688272  CAPLUS
DOCUMENT NUMBER:         133:280563
TITLE:                   Human antibodies that bind human IL-12 and methods for
                         producing
INVENTOR(S):             Salfeld, Jochen G.; Roguska, Michael; Paskind,
                         Michael; Banerjee, Subhashis; Tracey, Daniel E.;
                         White, Michael; Kaymakcalan, Zehra; Labkovsky, Boris;
                         Sakorafas, Paul; Friedrich, Stuart; Myles, Angela;
                         Veldman, Geertruida M.; Venturini, Amy; Warne,
                         Nicholas W.; Widom, Angela; Elvin, John G.; Duncan,
                         Alexander R.; Derbyshire, Elaine J.; Carmen, Sara;
                         Smith, Stephen; Holtet, Thor Las; Du, Fou Sarah L.
PATENT ASSIGNEE(S):      Basf A.-G., Germany; Genetics Institute Inc.; et al.
SOURCE:                  PCT Int. Appl., 377 pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

     PATENT NO.         KIND  DATE           APPLICATION NO.  DATE
     ---------------    ----  --------       ---------------  --------
     WO 2000056772      A1    20000928       WO 2000-US7946   20000324
         W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR,
            CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU,
            ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU,
            LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE,
            SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA,
            ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
            DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
            CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.:                  US 1999-126603   P  19990325
AB   Human antibodies, preferably recombinant human antibodies, that
     specifically bind to human interleukin-12 (hIL-12) are disclosed.
     Preferred antibodies have high affinity for hIL-12 and neutralize hIL-12
     activity in vitro and in vivo . An antibody of the invention can be a
     full-length antibody or an antigen-binding portion thereof. The
     antibodies, or antibody portions, of the invention are useful for
     detecting hIL-12 and for inhibiting hIL-12 activity, e.g., in a human
     subject suffering from a disorder in which hIL-12 activity is detrimental.
     Nucleic acids, vectors and host cells for expressing the recombinant human
     antibodies of the invention, and methods of synthesizing the recombinant
     human antibodies, are also encompassed by the invention.
REFERENCE COUNT:         7
REFERENCE(S):            (2) Carter, R; HYBRIDOMA 1997, V16(4), P363 CAPLUS
                         (3) Genentech Inc; WO 9404679 A 1994 CAPLUS
                         (4) Genetics Inst; WO 9524918 A 1995 CAPLUS
                         (5) Irving, R; IMMUNOTECHNOLOGY 1996, V2(2), P127
                             CAPLUS
                         (6) Pini, A; JOURNAL OF IMMUNOLOGICAL METHODS 1997,
                             V206(1-2), P171 CAPLUS
                        ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> s IL12 (10N) monoclonal
L2           1 IL12 (10N) MONOCLONAL

=> s ((IL12 or IL-12 or (Interleukin (1N) 12) or (Interleukin-12)) (4N) receptor) (10N) monoclonal
L3          20 ((IL12 OR IL-12 OR (INTERLEUKIN (1N) 12) OR (INTERLEUKIN-12))
             (4N) RECEPTOR) (10N) MONOCLONAL
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=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 14 DUP REM L3 (6 DUPLICATES REMOVED)

=> dis l4 ibib abs kwic 1-14

L4 ANSWER 1 OF 14 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001392813 MEDLINE
DOCUMENT NUMBER: 21340391 PubMed ID: 11447182
TITLE: Differential roles of interleukin-18 (IL-18) and IL12 for induction of gamma interferon by staphylococcal cell wall components and superantigens.
AUTHOR: Stuyt R J; Netea M G; Kim S H; Novick D; Rubinstein M; Kullberg B J; Van der Meer J W; Dinarello C A
CORPORATE SOURCE: Department of Medicine, University of Colorado Health Sciences Center, Denver, Colorado, USA.
CONTRACT NUMBER: AI-15614 (NIAID)
SOURCE: INFECTION AND IMMUNITY, (2001 Aug) 69 (8) 5025-30.
Journal code: GO7; 0246127. ISSN: 0019-9567.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010827
Last Updated on STN: 20010827
Entered Medline: 20010823
AB The roles of endogenous cytokines induced by either intact staphylococcal microorganisms or staphylococcal exotoxins were examined using human whole-blood cultures. To accomplish this, interleukin-18 binding protein (IL-18BP) and tumor necrosis factor binding protein (TNFbp) were used to neutralize IL-18 and TNF, respectively, whereas an anti-IL-12 **monoclonal** antibody was used to neutralize **IL-12** and the IL-1 **receptor** antagonist (IL-1Ra) was used to block IL-1 receptors. Heat-killed Staphylococcus epidermidis and Staphylococcus aureus, as well as the staphylococcal superantigens toxic shock syndrome toxin-1 (TSST-1) and staphylococcus enterotoxin B (SEB) induced gamma interferon (IFN-gamma) production. Staphylococcus spp.-induced production of IFN-gamma required the presence of endogenous IL-18, IL-12, and TNF. In contrast, TSST-1-induced IFN-gamma was not significantly reduced in the presence of IL-18BP, anti-IL-12 antibodies, IL-1Ra, or anti-TNFbp. SEB-induced IFN-gamma was significantly inhibited only by anti-IL-12 antibodies, indicating that endogenous IL-18, IL-1, and TNF are not required for SEB-induced IFN-gamma. In conclusion, the mechanisms of IFN-gamma stimulation by intact staphylococcal microorganisms and by exotoxins differ, and this is likely due to the different receptors which are triggered on the cell membranes. In contrast to its role in the interactions between staphylococci and host cells, IL-18 does not appear to play a major role in superantigen-induced IFN-gamma.
AB . . . protein (IL-18BP) and tumor necrosis factor binding protein (TNFbp) were used to neutralize IL-18 and TNF, respectively, whereas an anti-IL-12 **monoclonal** antibody was used to neutralize **IL -12** and the IL-1 **receptor** antagonist (IL-1Ra) was used to block IL-1 receptors. Heat-killed Staphylococcus epidermidis and Staphylococcus aureus, as well as the staphylococcal superantigens. . .

L4 ANSWER 2 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:456576 CAPLUS
DOCUMENT NUMBER: 135:179506
TITLE: Attenuation of bleomycin-induced pneumopathy in mice by monoclonal antibody to interleukin-12
AUTHOR(S): Maeyama, Takashige; Kuwano, Kazuyoshi; Kawasaki, Masayuki; Kunitake, Ritsuko; Hagimoto, Naoki; Hara, Nobuyuki
CORPORATE SOURCE: Research Institute for Diseases of the Chest, Graduate School of Medical Sciences, Kyushu University, Fukuoka, 812-8582, Japan
SOURCE: Am. J. Physiol. (2001), 280(6, Pt. 1), L1128-L1137
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We previously demonstrated essential roles of the Fas-Fas ligand (FasL) pathway in bleomycin-induced pneumopathy in mice. T lymphocytes and natural killer cells express FasL on activation and use it as a cytotoxic effector mol. Because interleukin (IL)-12 is known to play a crit. role in cell-mediated immunity, we investigated whether anti-IL-12 antibody treatment suppresses the development of this model. The anti-IL-12 antibody treatment decreased the no. of apoptotic cells and the degree of inflammation and fibrosis in lung tissue. The results of RT-PCR showed that IL-12p40, IL-12 receptor (R) .beta.2, interferon-.gamma., tumor necrosis factor-.alpha. and FasL mRNAs were upregulated after bleomycin instillation. The upregulation of FasL, IL-12R.beta.2, and tumor necrosis factor-.alpha. mRNA expression in lung tissue was suppressed by anti-IL-12 antibody treatment. The results of ELISA showed that the levels of IL-12p40, but not of IL-12p70, were increased in lung tissue after bleomycin instillation. Although the increase in IL-12R.beta.2 mRNA levels suggests that the T helper type 1 cell response may participate in lung injury, the increase in IL-12p40 supports T helper type 2 cell predominance in the fibrotic process of this model. The administration of anti-IL-12 antibody could be a novel therapy against lung injury and pulmonary fibrosis.
REFERENCE COUNT: 40
REFERENCE(S): (1) Bienkowski, R; Proc Soc Exp Biol Med 1995, V209, P118 CAPLUS
(2) Buttner, C; Am J Respir Cell Mol Biol 1997, V17, P315 CAPLUS
(3) Chandler, D; Am J Pathol 1983, V112, P170 CAPLUS
(4) Chen, L; J Immunol 1997, V159, P351 CAPLUS
(5) D'Andrea, A; J Exp Med 1992, V176, P1387 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT
IT Interleukin **receptors**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**interleukin 12**; attenuation of pneumopathy in mice by **monoclonal** antibody to interleukin-12 in relation to)

L4 ANSWER 3 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:688272 CAPLUS
DOCUMENT NUMBER: 133:280563
TITLE: Human antibodies that bind human IL-12 and methods for producing
INVENTOR(S): Salfeld, Jochen G.; Roguska, Michael; Paskind,

Michael; Banerjee, Sub[redacted]s; Tracey, Daniel E.; White, Michael; Kaymakcalan, Zehra; Labkovsky, Boris; Sakorafas, Paul; Friedrich, Stuart; Myles, Angela; Veldman, Geertruida M.; Venturini, Amy; Warne, Nicholas W.; Widom, Angela; Elvin, John G.; Duncan, Alexander R.; Derbyshire, Elaine J.; Carmen, Sara; Smith, Stephen; Holtet, Thor Las; Du, Fou Sarah L.
PATENT ASSIGNEE(S): Basf A.-G., Germany; Genetics Institute Inc.; et al.
SOURCE: PCT Int. Appl., 377 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000056772	A1	20000928	WO 2000-US7946	20000324

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-126603 P 19990325
AB Human antibodies, preferably recombinant human antibodies, that specifically bind to human interleukin-12 (hIL-12) are disclosed. Preferred antibodies have high affinity for hIL-12 and neutralize hIL-12 activity in vitro and in vivo . An antibody of the invention can be a full-length antibody or an antigen-binding portion thereof. The antibodies, or antibody portions, of the invention are useful for detecting hIL-12 and for inhibiting hIL-12 activity, e.g., in a human subject suffering from a disorder in which hIL-12 activity is detrimental. Nucleic acids, vectors and host cells for expressing the recombinant human antibodies of the invention, and methods of synthesizing the recombinant human antibodies, are also encompassed by the invention.
REFERENCE COUNT: 7
REFERENCE(S): (2) Carter, R; HYBRIDOMA 1997, V16(4), P363 CAPLUS
(3) Genentech Inc; WO 9404679 A 1994 CAPLUS
(4) Genetics Inst; WO 9524918 A 1995 CAPLUS
(5) Irving, R; IMMUNOTECHNOLOGY 1996, V2(2), P127 CAPLUS
(6) Pini, A; JOURNAL OF IMMUNOLOGICAL METHODS 1997, V206(1-2), P171 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 4 OF 14 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 2000:440409 BIOSIS
DOCUMENT NUMBER: PREV200000440409
TITLE: Antibody to IL-12 receptor.
AUTHOR(S): Chizzonite, Richard Anthony (1); Truitt, Theresa Patricia
CORPORATE SOURCE: (1) South Kent, CT USA
ASSIGNEE: Hoffmann-La Roche Inc.
PATENT INFORMATION: US 6046012 April 04, 2000
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Apr. 4, 2000) Vol. 1233, No. 1, pp. No pagination. e-file.
ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
AB This disclosure relates to novel antibodies specific to the recently discovered **receptor** to human **interleukin 12** (IL-12R). The antibodies to IL-12R, most preferably, the **monoclonal** antibodies to that protein, are useful in determining the status of the human immune system and as diagnostic reagents or potential therapeutic reagents for conditions involving imbalances in IL-12 levels or cell types sensitive to IL-12 activation. Further aspects of the disclosure relate to methods of producing and purifying such novel antibodies and hybridoma cell lines capable of their production. Another aspect of the disclosure relates to an immunoprecipitation assay for the detection of solubilized IL-12R which employs, in a preferred embodiment, monoclonal antibodies to the receptor of the present invention covalently bound to Protein G-Sepharose resin.
AB This disclosure relates to novel antibodies specific to the recently discovered **receptor** to human **interleukin 12** (IL-12R). The antibodies to IL-12R, most preferably, the **monoclonal** antibodies to that protein, are useful in determining the status of the human immune system and as diagnostic reagents or. . .

L4 ANSWER 5 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:350691 CAPLUS
DOCUMENT NUMBER: 130:351231
TITLE: **Monoclonal** antibody to the **interleukin-12 receptor** .beta.2-chain
INVENTOR(S): De Boer, Mark; Den Hartog, Marcel Theodorus
PATENT ASSIGNEE(S): Tanox Pharma B.V., Neth.
SOURCE: PCT Int. Appl., 24 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9925737	A1	19990527	WO 1998-NL663	19981119

W: CA, JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

PRIORITY APPLN. INFO.: EP 1997-203607 19971119
AB The authors disclose the prepn. of a **monoclonal** antibody (3H4) that binds to the **interleukin-12 receptor** (IL-12R) .beta.2 chain expressed on the cell surface of human T lymphocytes. Binding of this monoclonal antibody prevents IL-12R .beta.2 chain-mediated STAT4 phosphorylation. The authors suggest this antibody may be combined with autoantigens or with other antibodies to co-stimulatory receptors on T cells or antigen presenting cells in therapy of autoimmune diseases.
REFERENCE COUNT: 7

REFERENCE(S): (1) BASF Aktiengesells[redacted]; WO 9841232 A 1998 CAPLUS
(2) F Hoffman La Roche AG; EP 0638644 A 1995 CAPLUS
(3) F Hoffman La Roche AG; EP 0759466 A 1997 CAPLUS
(4) Hoffman-La Roche Inc; US 5852176 A 1998 CAPLUS
(5) Hoffman-La Roche Inc; US 5853721 A 1998 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI **Monoclonal** antibody to the **interleukin-12 receptor** .beta.2-chain

AB The authors disclose the prepn. of a **monoclonal** antibody (3H4) that binds to the **interleukin-12 receptor** (IL-12R) .beta.2 chain expressed on the cell surface of human T lymphocytes. Binding of this monoclonal antibody prevents IL-12R .beta.2 chain-mediated STAT4 phosphorylation. The authors suggest this antibody may be combined with autoantigens or with other antibodies to co-stimulatory receptors on T cells or antigen presenting cells in therapy of autoimmune diseases.

ST autoimmune disease **monoclonal** antibody **interleukin 12 receptor** beta2 chain

IT **Interleukin receptors**
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(12; **monoclonal** antibody to .beta.2 chain of)

IT STAT transcription factors
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(STAT4 transcription factor; **monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain antagonizes signal transduction-induced phosphorylation of)

IT Spectrins
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(fodrins, .alpha.-; **monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain in therapeutic combination with)

IT Glycoproteins (specific proteins and subclasses)
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gp-39; **monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain in therapeutic combination with)

IT Molecular association
(**monoclonal** antibody antagonism of **interleukin-12 receptor** .beta.2 chain dimerization with .beta.1 chain)

IT T cell (lymphocyte)
Th1 cell
(**monoclonal** antibody antagonism of **interleukin-12 receptor** .beta.2 chain dimerization with .beta.1 chain and receptor-mediated signal transduction in)

IT Protein phosphorylation
Signal transduction (biological)
(**monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain antagonizes signal transduction-induced phosphorylation of STAT4)

IT Antidiabetic agents
Antirheumatic drugs
(**monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain in)

IT Th2 cell
(**monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain in combination with heat shock proteins for stimulation of type 2 cytokine secretion by)

IT Autoantigens
Heat-shock proteins
Myelin basic protein
Type II collagen
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain in therapeutic combination with)

IT Autoimmune diseases
Sjogren's syndrome
(**monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain in therapy of)

IT CD40 (antigen)
CD40 ligand
CD80 (antigen)
CD86 (antigen)
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain in therapy of autoimmune disease in combination with monoclonal antibody to)

IT Antigen-presenting cell
(**monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain in therapy of autoimmune disease in combination with monoclonal antibody to T-cell co-stimulatory receptor on)

IT **Interleukin 12**
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(**receptors**; **monoclonal** antibody to .beta.2 chain of)

IT Monoclonal antibodies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(to T-cell co-stimulatory receptors in combination therapy with **monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain)

IT **Monoclonal** IgG1
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(to **interleukin-12 receptor** .beta.2 chain and antagonistic for signal transduction)

IT Immunotherapy
(with autoantigens and **monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain)

IT 9004-10-8, Insulin, biological studies 9024-58-2, Glutamate decarboxylase
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**monoclonal** antibody to **interleukin-12 receptor** .beta.2 chain in therapeutic combination with)

L4 ANSWER 6 OF 14 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000056353 MEDLINE
DOCUMENT NUMBER: 20056353 PubMed ID: 10588628
TITLE: DNA from Mycobacterium bovis bacillus Calmette-Guerin (MY-1) inhibits immunoglobulin E production by human lymphocytes.
AUTHOR: Fujieda S; Iho S; Kimura Y; Sunaga H; Igawa H; Sugimoto C; Yamamoto S; Saito H

CORPORATE SOURCE: Departments of Otorhinolaryngology and Immunology, Fukui Medical University, Fukui, Matsuoka, Yoshida, Japan.. sfujieda@fmsrsa.fukui-med.ac.jp
SOURCE: AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE, (1999 Dec) 160 (6) 2056-61.
Journal code: BZS; 9421642. ISSN: 1073-449X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000413
Last Updated on STN: 20000413
Entered Medline: 20000403

AB A DNA fraction purified from Mycobacterium bovis bacillus Calmette-Guerin (BCG) and designated MY-1 induced interferon (IFN)-gamma production by human peripheral blood mononuclear cells (PBMC). IFN-gamma is well known as a downregulator of IgE production. In this study we investigated whether MY-1 regulates IgE production by human PBMC in vitro. MY-1 inhibited IgE production in PBMC taken from normal donors and stimulated with interleukin (IL)-4 plus monoclonal anti-CD40 antibody, without affecting production of IgA. MY-1 enhanced production of IFN-gamma and IL-12 by PBMC. Inhibition by MY-1 of IgE production was mediated by both IFN-gamma and IL-12, since the MY-1-induced suppression was blocked by the addition of monoclonal anti-IFN-gamma antibody, monoclonal anti-IL-12 antibody or a **monoclonal** antibody (mAb) directed at the **IL-12 receptor**. MY-1 inhibited the induction of epsilon germ-line transcript by IL-4. Additionally, MY-1 inhibited spontaneous in vitro production of IgE by PBMC from atopic donors in the absence of IL-4 plus anti-CD40 mAb. These results suggest that exposure to MY-1 may be a novel strategy for the treatment of IgE-related allergic disease.

AB . . . and IL-12, since the MY-1-induced suppression was blocked by the addition of monoclonal anti-IFN-gamma antibody, monoclonal anti-IL-12 antibody or a **monoclonal** antibody (mAb) directed at the **IL-12 receptor**. MY-1 inhibited the induction of epsilon germ-line transcript by IL-4. Additionally, MY-1 inhibited spontaneous in vitro production of IgE by. . .

L4 ANSWER 7 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:706111 CAPLUS
DOCUMENT NUMBER: 129:314955
TITLE: Inhibition of B-1 cell mediated immune conditions
INVENTOR(S): Askenase, Philip W.; Tsuji, Ryohei; Paliwal, Vipin; Kawikova, Ivana
PATENT ASSIGNEE(S): Yale University, USA
SOURCE: PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9846255	A1	19981022	WO 1998-US7535	19980417
W: AU, CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9871180	A1	19981111	AU 1998-71180	19980417
PRIORITY APPLN. INFO.:			US 1997-45234	19970417
			WO 1998-US7535	19980417

AB This invention relates to methods of identifying agents that can inhibit delayed type hypersensitivity (DTH) reactions within the first few hours of exposure to an antigen or allergen that can trigger a DTH response. The invention also discloses methods of preventing DTH and contact sensitivity (CS) responses by preventing activation of the classical complement cascade through the modulation of IgM antibodies which are synthesized by B-1 (CD5+) type B cells. The invention also discloses methods of identifying agents that inhibit the hypersensitivity response by inhibiting prodn. of the B-1 cell DTH-initiating IgM antibody, or by inhibiting DTH-initiating IgM antibody activation of the classical complement cascade.

ST B1 cell delayed type hypersensitivity inhibitor; **monoclonal** antibody C5a **receptor interleukin 12**

L4 ANSWER 8 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:21581 CAPLUS
DOCUMENT NUMBER: 130:80350
TITLE: Antibody to interleukin-12 receptor
INVENTOR(S): Gately, Maurice Kent; Presky, David Howard; Wu, Chang-You
PATENT ASSIGNEE(S): Hoffmann-La Roche Inc., USA
SOURCE: U.S., 35 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5853721	A	19981229	US 1995-381059	19950131

AB The present invention relates to a novel antibody against the IL-12 receptor and a novel combination of antibodies against the IL-12 receptor. The novel anti-IL-12 receptor antibody, designated as 2B10, provided in accordance with the present invention binds to the human IL-12 receptor but which is not capable of inhibiting the binding of human IL-12 to the high affinity human IL-12 receptor and is not capable of neutralizing human IL-12 bioactivity by binding to human IL-12 receptor. Combination of these antibodies inhibit IL-12-induced proliferation of activated T cells, reduce IL-12-induced secretion of interferon .gamma. by resting peripheral blood mononuclear cells, and suppress IL-12-induced activation of lymphokine-activated killer cells. These antibodies are therefore useful for therapeutic intervention in septic shock, autoimmune disease, multiple sclerosis, and rheumatoid arthritis.

REFERENCE COUNT: 15
REFERENCE(S): (1) Anon; EP 239400 1987 CAPLUS
(2) Anon; WO 92/11018 1992 CAPLUS
(3) Chan; J Exp Med 1991, V173, P869 CAPLUS
(4) Chizzonite, R; J Immunol 1991, V147, P1548 CAPLUS
(5) Chizzonite, R; J Immunol 1992, V148, P3117 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

IT **Monoclonal** antibodies
RL: BPN (Biosynthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(antibody to **interleukin-12 receptor** for treating septic shock and autoimmune disease)

L4 ANSWER 9 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:696638 CAPLUS
DOCUMENT NUMBER: 128:727
TITLE: DHEA combination therapy with interleukin antibodies for antiviral, antibacterial, antimycoplasmal, or anti-intracellular parasite therapy
INVENTOR(S): Prendergast, Patrick T.
PATENT ASSIGNEE(S): Prendergast, Patrick T., Ire.
SOURCE: PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9738695	A1	19971023	WO 1997-IB414	19970417
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2251733	AA	19971023	CA 1997-2251733	19970417
AU 9725741	A1	19971107	AU 1997-25741	19970417
AU 734807	B2	20010621		
EP 901375	A1	19990317	EP 1997-917365	19970417
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
CN 1216470	A	19990512	CN 1997-193912	19970417
JP 2000508654	T2	20000711	JP 1997-536909	19970417
WO 9847516	A1	19981029	WO 1997-EP5716	19971016
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9852219	A1	19981113	AU 1998-52219	19971016
NO 9804851	A	19981217	NO 1998-4851	19981016

PRIORITY APPLN. INFO.:
US 1996-15695 P 19960417
WO 1970-IB414 A 19970417
WO 1997-IB414 W 19970417
WO 1997-EP5716 W 19971016

OTHER SOURCE(S): MARPAT 128:727

AB There are provided medicaments, methods of making them, and kits, which include (1) a 17-ketosteroid compd. and/or (2) anti-serum either poly- or monoclonal to Interleukin 10, Interleukin 2, or Interleukin 12, or with any compd. which can effectively inhibit synthesis or the biol. function of Interleukin 10, Interleukin 12, or Interleukin 2, or with an Interleukin 10, **Interleukin 12**, or **Interleukin 2 receptor** mol.-blocking agent, or with anti-serum, either polyclonal or **monoclonal** to human .alpha.-fetoprotein. There are also provided methods of treatment involving such compds. or combinations of compds., including enhancing Th1 immune protective responses when using the 17-ketosteroid compd. as an anti-viral, anti-bacterial, anti-mycoplasm or anti-intracellular parasitic agent.
AB There are provided medicaments, methods of making them, and kits, which include (1) a 17-ketosteroid compd. and/or (2) anti-serum either poly- or monoclonal to Interleukin 10, Interleukin 2, or Interleukin 12, or with any compd. which can effectively inhibit synthesis or the biol. function of Interleukin 10, Interleukin 12, or Interleukin 2, or with an Interleukin 10, **Interleukin 12**, or **Interleukin 2 receptor** mol.-blocking agent, or with anti-serum, either polyclonal or **monoclonal** to human .alpha.-fetoprotein. There are also provided methods of treatment involving such compds. or combinations of compds., including enhancing Th1 immune protective responses when using the 17-ketosteroid compd. as an anti-viral, anti-bacterial, anti-mycoplasm or anti-intracellular parasitic agent.

L4 ANSWER 10 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:90070 CAPLUS
DOCUMENT NUMBER: 126:292260
TITLE: Regulation of interleukin-12 receptor .beta.1 chain expression and interleukin-12 binding by human peripheral blood mononuclear cells
AUTHOR(S): Wu, Chang You; Warrier, Rajeev R.; Wang, Xin; Presky, David H.; Gately, Maurice K.
CORPORATE SOURCE: Dep. Inflammation/Autoimmune Diseases, Hoffmann-La Roche Inc., Nutley, NJ, 07110, USA
SOURCE: Eur. J. Immunol. (1997), 27(1), 147-154
CODEN: EJIMAF; ISSN: 0014-2980
PUBLISHER: VCH
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Activation of human peripheral blood mononuclear cells (PBMC) with anti-CD3 **monoclonal** antibody (mAb) or phytohemagglutinin resulted in the up-regulation of **interleukin-12 receptor** (IL-12R).beta.1 expression and IL-12 binding. Kinetic studies revealed that max. expression of IL-12R.beta.1 and IL-12 binding occurred on days 3-4. Anti-CD3-induced expression of IL-12R.beta.1 chain and IL-12 binding by PBMC was augmented by anti-CD28 mAb, indicating that the potentiating effect of anti-CD28 on T cell responses to IL-12 could be mediated, at least in part, by the enhancement of IL-12R expression. Among 16 cytokines, IL-2, IL-7, and IL-15 markedly induced IL-12R.beta.1 expression and IL-12 binding on resting PBMC, whereas IL-1.alpha. and tumor necrosis factor-.alpha. had a minimal enhancing effect. IL-3, IL-4, IL-S, IL-6, IL-8, IL-10, IL-12, interferon (IFN)-.alpha., IFN-.gamma., granulocyte/macrophage colony-stimulating factor, and transforming growth factor (TGF)-.beta.2 had no detectable enhancing effect. Anti-CD3-induced expression of IL-12R.beta.1 and of low-affinity IL-12 binding sites was partially inhibited by TGF-.beta.2, IL-10 and IL-4; TGF-.beta.2 and IL-10

completely abolished anti-CD3-induced expression of high-affinity IL-12 binding sites. Consistent with the redn. of high affinity IL-12 binding sites, PBMC activated with anti-CD3 mAb in the presence of TGF-.beta.2 or IL-10 failed to produce IFN-.gamma. or to proliferate in response to IL-12. It was suggested that Th2 cell-derived cytokines can inhibit IL-12-induced biol. functions by inhibiting IL-12R expression and that expression of a second subunit of the IL-12R (IL-12R.beta.2), required for the formation of high-affinity IL-12 binding sites, may he more highly regulated by TGF-.beta.2 and IL-10 than is expression of IL-12R.beta.1.

AB Activation of human peripheral blood mononuclear cells (PBMC) with anti-CD3 **monoclonal** antibody (mAb) or phytohemagglutinin resulted in the up-regulation of **interleukin-12 receptor** (IL-12R).beta.1 expression and IL-12 binding. Kinetic studies revealed that max. expression of IL-12R.beta.1 and IL-12 binding occurred on days 3-4. Anti-CD3-induced expression of IL-12R.beta.1 chain and IL-12 binding by PBMC was augmented by anti-CD28 mAb, indicating that the potentiating effect of anti-CD28 on T cell responses to IL-12 could be mediated, at least in part, by the enhancement of IL-12R expression. Among 16 cytokines, IL-2, IL-7, and IL-15 markedly induced IL-12R.beta.1 expression and IL-12 binding on resting PBMC, whereas IL-1.alpha. and tumor necrosis factor-.alpha. had a minimal enhancing effect. IL-3, IL-4, IL-S, IL-6, IL-8, IL-10, IL-12, interferon (IFN)-.alpha., IFN-.gamma., granulocyte/macrophage colony-stimulating factor, and transforming growth factor (TGF)-.beta.2 had no detectable enhancing effect. Anti-CD3-induced expression of IL-12R.beta.1 and of low-affinity IL-12 binding sites was partially inhibited by TGF-.beta.2, IL-10 and IL-4; TGF-.beta.2 and IL-10 completely abolished anti-CD3-induced expression of high-affinity IL-12 binding sites. Consistent with the redn. of high affinity IL-12 binding sites, PBMC activated with anti-CD3 mAb in the presence of TGF-.beta.2 or IL-10 failed to produce IFN-.gamma. or to proliferate in response to IL-12. It was suggested that Th2 cell-derived cytokines can inhibit IL-12-induced biol. functions by inhibiting IL-12R expression and that expression of a second subunit of the IL-12R (IL-12R.beta.2), required for the formation of high-affinity IL-12 binding sites, may he more highly regulated by TGF-.beta.2 and IL-10 than is expression of IL-12R.beta.1.

L4 ANSWER 11 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:467405 CAPLUS
DOCUMENT NUMBER: 125:112774
TITLE: Recombinant DNA encoding human receptor for interleukin-12
INVENTOR(S): Chua, Anne O.; Gubler, Ulrich A.
PATENT ASSIGNEE(S): Hoffmann-La Roche Inc., USA
SOURCE: U.S., 47 pp. Cont.-in-part of U.S. Ser. No. 94, 713, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5536657	A	19960716	US 1994-248532	19940531
EP 638644	A1	19950215	EP 1994-110657	19940708
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
CA 2128151	AA	19950120	CA 1994-2128151	19940715
AU 9467505	A1	19950127	AU 1994-67505	19940715
AU 676325	B2	19970306		
JP 07194383	A2	19950801	JP 1994-166950	19940719
US 5831007	A	19981103	US 1995-419652	19950411

PRIORITY APPLN. INFO.: US 1993-94713 19930719
US 1993-94649 19930719
US 1994-248532 19940531

AB Low-affinity receptors for interleukin-12 are identified in human and cDNAs encoding them are cloned and antibodies raised against the receptors. The receptors bind interleukin-12 in a specific and saturable manner with an apparent affinity KD of .apprx.2-10 nM. The interleukin-12 receptor is shown to be a member of the cytokine receptor superfamily and has a high homol. to human gp130. The receptors are 662 and 660 amino acid residues in length with a 23-residue signal moiety, and differ only slightly at the C-terminus, probably as a result of alternative splicing. Prepn. of monoclonal antibodies to the receptors and their use in the characterization of the receptor and in the cloning of the cDNAs are described.

IT Antibodies
RL: BPN (Biosynthetic preparation); BUU (Biological use, unclassified); BIOL (Biological study); PREP (Preparation); USES (Uses)
(**monoclonal**, recombinant DNA encoding human **receptor** for **interleukin-12**)

L4 ANSWER 12 OF 14 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97118032 MEDLINE
DOCUMENT NUMBER: 97118032 PubMed ID: 8958915
TITLE: Molecular biology of interleukin-12 receptors.
AUTHOR: Gubler U; Presky D H
CORPORATE SOURCE: Department of Inflammation/Autoimmune Diseases Hoffmann-La Roche Inc., Nutley, New Jersey 07110, USA.
SOURCE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1996 Oct 31) 795 36-40.
Journal code: 5NM; 7506858. ISSN: 0077-8923.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199701
ENTRY DATE: Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19970108

AB IL-12 receptors are expressed primarily on activated T and NK cells. Using labeled IL-12, three classes of IL-12-binding sites have been identified on human PHA-activated lymphoblasts. Their Kd values are 5-20 pM (high affinity), 50-200 pM (intermediate affinity), and 2-6 nM (low affinity). Using a **monoclonal** antibody, a cDNA encoding a human **IL -12 receptor** was isolated by expression cloning. The homologous mouse cDNA was isolated by cross hybridization. These proteins are gp130-like members of the cytokine receptor superfamily. Individually, however they bind IL-12 with low affinity. An expression cloning approach was undertaken to isolate an additional human IL-12 receptor component that would act as a high-affinity converter. A cDNA encoding another IL-12 receptor was isolated. The mouse homologue was obtained by cross hybridization. These IL-12 receptors are also gp130-like cytokine receptor superfamily members. Because these two types of IL-12 receptors have the

general makeup of beta-type cytokine recep[redacted]subunits, they are designated as IL-12R beta 1 and IL-12R beta 2. Coexpression of IL-12R beta 1 and IL-12R beta 2 leads to the formation of high-affinity IL-12-binding sites and reconstitution of IL-12-dependent signaling.

AB . . . lymphoblasts. Their Kd values are 5-20 pM (high affinity), 50-200 pM (intermediate affinity), and 2-6 nM (low affinity). Using a **monoclonal** antibody, a cDNA encoding a human **IL-12 receptor** was isolated by expression cloning. The homologous mouse cDNA was isolated by cross hybridization. These proteins are gp130-like members of. . .

L4 ANSWER 13 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:573824 CAPLUS
DOCUMENT NUMBER: 123:7879
TITLE: Interleukin-12 receptors with low affinity and cDNAs encoding them and antibodies to the receptors
INVENTOR(S): Chizzonite, Richard Anthony; Chua, Anne On; Gubler, Ulrich Andreas; Truitt, Theresa Patricia
PATENT ASSIGNEE(S): F. Hoffmann-La Roche AG, Switz.
SOURCE: Eur. Pat. Appl., 61 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 638644	A1	19950215	EP 1994-110657	19940708
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
US 5536657	A	19960716	US 1994-248532	19940531
ZA 9405154	A	19950119	ZA 1994-5154	19940714
US 6046012	A	20000404	US 1997-789350	19970127

PRIORITY APPLN. INFO.: US 1993-94649 19930719
US 1993-94713 19930719
US 1994-248532 19940531
US 1994-248531 19940531

AB Low-affinity receptors for interleukin-12 are identified in human and mouse and cDNAs encoding them are cloned and antibodies raised against the receptors. The receptor has a KD for interleukin 12 of <10 nM. Prepn. of monoclonal antibodies to the receptor and their use in the characterization of the receptor and in the cloning of the cDNAs are described.

ST interleukin 12 receptor cDNA mouse human; antibody **monoclonal** **interleukin 12 receptor**

IT Antibodies
RL: MSC (Miscellaneous)
(**monoclonal**, to low-affinity **interleukin-12 receptors**; **interleukin-12 receptors** with low affinity and cDNAs encoding them and antibodies to receptors)

L4 ANSWER 14 OF 14 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:653750 CAPLUS
DOCUMENT NUMBER: 115:253750
TITLE: IL-12: monoclonal antibodies specific for the 40kDa subunit block receptor binding and biologic activity on activated human lymphoblasts
AUTHOR(S): Chizzonite, Richard; Truitt, Terri; Podlaski, Frank J.; Wolitzky, Aimee G.; Quinn, Phyllis M.; Nunes, Perla; Stern, Alvin S.; Gately, Maurice K.
CORPORATE SOURCE: Roche Res. Cent., Hoffmann-La Roche, Inc., Nutley, NJ, 07110, USA
SOURCE: J. Immunol. (1991), 147(5), 1548-56
CODEN: JOIMA3; ISSN: 0022-1767
DOCUMENT TYPE: Journal
LANGUAGE: English

AB This report describes the development of anti-IL-12 mAb that are specific for the 40-kDa subunit. Two classes of antibodies are characterized: inhibitory antibodies that block IL-12 binding to its cellular receptor and neutralize IL-12 bioactivity and noninhibitory antibodies that bind IL-12 without blocking receptor binding or neutralizing bioactivity.

IT **Receptors**
RL: BIOL (Biological study)
(for **interleukin 12**, on activated human lymphoblasts, ligand binding to, **monoclonal** antibody inhibition of)

IT Lymphoblast
(**interleukin 12 receptors** on human, ligand -binding to, **monoclonal** antibody inhibition of)

=> s de boer M?/au or Den Harog M?/au
L5 1001 DE BOER M?/AU OR DEN HAROG M?/AU

=> s l5 IL12
MISSING OPERATOR L5 IL12
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l5 and IL12
L6 0 L5 AND IL12

=> s l5 and IL-12
L7 18 L5 AND IL-12

=> s l5 and (IL-12 (5N) receptor)
L8 0 L5 AND (IL-12 (5N) RECEPTOR)

=> dup rem l7
PROCESSING COMPLETED FOR L7
L9 5 DUP REM L7 (13 DUPLICATES REMOVED)

=> dis l9 1-5 ibib abs kwic

L9 ANSWER 1 OF 5 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000281694 MEDLINE
DOCUMENT NUMBER: 20281694 PubMed ID: 10820284
TITLE: Prevention of experimental colitis in SCID mice reconstituted with CD45RBhigh CD4+ T cells by blocking the CD40-CD154 interactions.
AUTHOR: Liu Z; Geboes K; Colpaert S; Overbergh L; Mathieu C; Heremans H; **de Boer M**; Boon L; D'Haens G; Rutgeerts P; Ceuppens J L

CORPORATE SOURCE: Laboratory of Experimental [redacted]nology, Department of Pathology, University Hospital Gasthuisberg, Leuven, Belgium.
SOURCE: JOURNAL OF IMMUNOLOGY, (2000 Jun 1) 164 (11) 6005-14. Journal code: IFB; 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200006
ENTRY DATE: Entered STN: 20000629
Last Updated on STN: 20000629
Entered Medline: 20000621

AB Increased expression of CD40 and CD40 ligand (CD40L or CD154) has been found in inflamed mucosa of human inflammatory bowel disease (IBD), and interactions between these molecules seem to be involved in local cytokine production by macrophages. However, the precise role of CD40 signaling in the pathogenesis of IBD is still poorly understood. The aim of the present study was to investigate the in vivo relevance of CD40 signaling in experimental colitis in SCID mice reconstituted with syngeneic CD45RBhighCD4+ T cells. The results demonstrated that CD40+ and CD40L+ cells as well as their mRNA levels were significantly increased in inflamed mucosa. Administration of anti-CD40L neutralizing mAb over an 8-wk period starting immediately after CD45RBhighCD4+ T cell reconstitution completely prevented symptoms of wasting disease. Intestinal mucosal inflammation was effectively prevented, as revealed by abrogated leukocyte infiltration and decreased CD54 expression and strongly diminished mRNA levels of the proinflammatory cytokines IFN-gamma, TNF, and IL-12. When colitic SCID mice were treated with anti-CD40L starting at 5 wk after T cell transfer up to 8 wk, this delayed treatment still led to significant clinical and histological improvement and down-regulated proinflammatory cytokine secretion. These data suggest that the CD40-CD40L interactions are essential for the Th1 inflammatory responses in the bowel in this experimental model of colitis. Blockade of CD40 signaling may be beneficial to human IBD.

AU Liu Z; Geboes K; Colpaert S; Overbergh L; Mathieu C; Heremans H; **de Boer M**; Boon L; D'Haens G; Rutgeerts P; Ceuppens J L

AB . . . by abrogated leukocyte infiltration and decreased CD54 expression and strongly diminished mRNA levels of the proinflammatory cytokines IFN-gamma, TNF, and **IL-12**. When colitic SCID mice were treated with anti-CD40L starting at 5 wk after T cell transfer up to 8 wk,. . .

L9 ANSWER 2 OF 5 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999421862 MEDLINE
DOCUMENT NUMBER: 99421862 PubMed ID: 10491009
TITLE: Hyperexpression of CD40 ligand (CD154) in inflammatory bowel disease and its contribution to pathogenic cytokine production.
AUTHOR: Liu Z; Colpaert S; D'Haens G R; Kasran A; **de Boer M**; Rutgeerts P; Geboes K; Ceuppens J L
CORPORATE SOURCE: Laboratory of Experimental Immunology, Department of Gastroenterology, University Hospital Gasthuisberg, Leuven, Belgium.
SOURCE: JOURNAL OF IMMUNOLOGY, (1999 Oct 1) 163 (7) 4049-57. Journal code: IFB; 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199910
ENTRY DATE: Entered STN: 19991101
Last Updated on STN: 19991101
Entered Medline: 19991021

AB CD40 ligand (CD40L or CD154), a type II membrane protein with homology to TNF, is transiently expressed on activated T cells and known to be important for B cell Ig production and for activation and differentiation of monocytes and dendritic cells. Both Crohn's disease and ulcerative colitis are characterized by local production of cytokines such as TNF and by an influx of activated lymphocytes into inflamed mucosa. Herein, we investigated whether CD40L signaling participates in immune responses in these diseases. Our results demonstrated that CD40L was expressed on freshly isolated lamina propria T cells from these patients and was functional to induce **IL-12** and TNF production by normal monocytes, especially after IFN-gamma priming. The inclusion of a blocking mAb to CD40L or CD40 in such cocultures significantly decreased monocyte **IL-12** and TNF production. Moreover, lamina propria and peripheral blood T cells from these patients, after in vitro activation with anti-CD3, showed increased and prolonged expression of CD40L as compared with controls. Immunohistochemical analyses indicated that the number of CD40+ and CD40L+ cells was significantly increased in inflamed mucosa, being B cells/macrophages and CD4+ T cells, respectively. These findings suggest that CD40L up-regulation is involved in pathogenic cytokine production in inflammatory bowel disease and that blockade of CD40-CD40L interactions may have therapeutic effects for these patients.

AU Liu Z; Colpaert S; D'Haens G R; Kasran A; **de Boer M**; Rutgeerts P; Geboes K; Ceuppens J L

AB . . . demonstrated that CD40L was expressed on freshly isolated lamina propria T cells from these patients and was functional to induce **IL-12** and TNF production by normal monocytes, especially after IFN-gamma priming. The inclusion of a blocking mAb to CD40L or CD40 in such cocultures significantly decreased monocyte **IL-12** and TNF production. Moreover, lamina propria and peripheral blood T cells from these patients, after in vitro activation with anti-CD3,. . .

L9 ANSWER 3 OF 5 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1998318176 MEDLINE
DOCUMENT NUMBER: 98318176 PubMed ID: 9655470
TITLE: Expression of accessory molecules and cytokines in acute EAE in marmoset monkeys (Callithrix jacchus).
AUTHOR: Laman J D; van Meurs M; Schellekens M M; **de Boer M**; Melchers B; Massacesi L; Lassmann H; Claassen E; Hart B A
CORPORATE SOURCE: Division of Immunological and Infectious Diseases, TNO Prevention and Health, Leiden, The Netherlands.. jd.laman@pg.tno.nl
SOURCE: JOURNAL OF NEUROIMMUNOLOGY, (1998 Jun 1) 86 (1) 30-45. Journal code: HSO; 8109498. ISSN: 0165-5728.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199807

ENTRY DATE: Entered STN: 19980723
Last Updated on STN: 20000922
Entered Medline: 19980716

AB Accessory molecules and cytokines are involved in the immunopathogenesis of multiple sclerosis (MS) and experimental autoimmune encephalomyelitis (EAE) in rodent models, and are potential targets for immunotherapy. Evaluation of such experimental therapies requires appropriate animal models. Therefore, we analysed the expression of selected accessory molecules and cytokines in the brain of marmoset monkeys (Callithrix jacchus) with acute EAE, a newly described non-human primate model for MS. All animals experienced active disease clinically and histopathologically with strong resemblance to MS. Perivascular infiltrates of mononuclear cells showed abundant expression of CD40. CD40 was expressed on macrophages, indicating that T cell priming and macrophage effector functions may result from local CD40-CD40L interactions. CD40 ligand (CD40L) and B7-2 (CD86) were also expressed, but to a lower extent, while B7-1 (CD80) expression was limited. Both pro-inflammatory and anti-inflammatory cytokines were produced within individual lesions during active disease (IFN-alpha, IFN-gamma, TNF-alpha, IL-1alpha, IL-1beta, IL-2, IL-4, IL-10 and **IL-12**). This suggests that relative levels rather than sequential expression of Th1- and Th2-type cytokines determine disease activity. These findings demonstrate the value of EAE in marmoset monkeys as a model to assess the role of accessory molecules and cytokines in multiple sclerosis, and to evaluate targeted intervention.

AU Laman J D; van Meurs M; Schellekens M M; **de Boer M**; Melchers B; Massacesi L; Lassmann H; Claassen E; Hart B A

AB . . . and anti-inflammatory cytokines were produced within individual lesions during active disease (IFN-alpha, IFN-gamma, TNF-alpha, IL-1alpha, IL-1beta, IL-2, IL-4, IL-10 and **IL-12**). This suggests that relative levels rather than sequential expression of Th1- and Th2-type cytokines determine disease activity. These findings demonstrate. . .

L9 ANSWER 4 OF 5 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 97436552 MEDLINE
DOCUMENT NUMBER: 97436552 PubMed ID: 9292525
TITLE: Human dendritic cells require exogenous interleukin-12-inducing factors to direct the development of naive T-helper cells toward the Th1 phenotype.
AUTHOR: Hilkens C M; Kalinski P; **de Boer M**; Kapsenberg M L
CORPORATE SOURCE: Academic Medical Center, University of Amsterdam, Department of Cell Biology & Histology, The Netherlands.
SOURCE: BLOOD, (1997 Sep 1) 90 (5) 1920-6.
Journal code: A8G; 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199709
ENTRY DATE: Entered STN: 19971013
Last Updated on STN: 19971013
Entered Medline: 19970930

AB Dendritic cells (DC) are important initiators of specific primary immune responses because they are the only APC that can efficiently activate naive Th cells. DC have the capacity to produce interleukin-12 (**IL-12**), a cytokine that plays a pivotal role in the development of Th1-mediated cellular immune responses. The present study focuses on the conditions under which human DC produce bioactive **IL-12** p70 and, consequently, direct the development of naive T helper (Th) cells toward the Th1 phenotype. Bacteria or bacterial compounds such as Staphylococcus aureus Cowan strain I (SAC) or lipopolysaccharide (LPS) induced substantial **IL-12** levels in DC, which could be further upregulated by interferon-gamma (IFN-gamma), whereas induction of **IL-12** production via CD40 ligation required IFN-gamma as an obligatory, complementary signal. Also, activated naive Th cells were poor inducers of **IL-12** production, unless exogenous IFN-gamma was present, whereas activated memory Th cells were effective inducers of **IL-12** production and did not require exogenous IFN-gamma. Next, the cytokine profiles of matured Th cells that were primed by DC under different conditions were examined. DC promoted the development of naive Th cells into memory Th0 cells that produced both the type 1 cytokine IFN-gamma and the type 2 cytokine IL-4. In contrast, after activation with SAC, DC efficiently directed the development of Th1 cells through the release of **IL-12**. An APC-independent Th cell maturation model, using either recombinant **IL-12** or supernatants of SAC-activated DC and neutralizing anti-**IL-12** antibodies, confirmed that DC-derived **IL-12** was the major Th1 skewing factor. Together, these data indicate that the contact between DC and naive Th cells during the initiation of specific immune responses does not result in the efficient induction of **IL-12** production and that, consequently, exogenous **IL-12**-inducing factors are required to promote primary Th1-mediated cellular immune responses.

AU Hilkens C M; Kalinski P; **de Boer M**; Kapsenberg M L

AB . . . because they are the only APC that can efficiently activate naive Th cells. DC have the capacity to produce interleukin-12 (**IL-12**), a cytokine that plays a pivotal role in the development of Th1-mediated cellular immune responses. The present study focuses on the conditions under which human DC produce bioactive **IL-12** p70 and, consequently, direct the development of naive T helper (Th) cells toward the Th1 phenotype. Bacteria or bacterial compounds such as Staphylococcus aureus Cowan strain I (SAC) or lipopolysaccharide (LPS) induced substantial **IL-12** levels in DC, which could be further upregulated by interferon-gamma (IFN-gamma), whereas induction of **IL-12** production via CD40 ligation required IFN-gamma as an obligatory, complementary signal. Also, activated naive Th cells were poor inducers of **IL-12** production, unless exogenous IFN-gamma was present, whereas activated memory Th cells were effective inducers of **IL-12** production and did not require exogenous IFN-gamma. Next, the cytokine profiles of matured Th cells that were primed by DC. . . cytokine IL-4. In contrast, after activation with SAC, DC efficiently directed the development of Th1 cells through the release of **IL-12**. An APC-independent Th cell maturation model, using either recombinant **IL-12** or supernatants of SAC-activated DC and neutralizing anti-**IL-12** antibodies, confirmed that DC-derived **IL-12** was the major Th1 skewing factor. Together, these data indicate that the contact between DC and naive Th cells during the initiation of specific immune responses does not result in the efficient induction of **IL-12** production and that, consequently, exogenous **IL-12**-inducing factors are required to promote primary Th1-mediated

cellular immune responses.

L9 ANSWER 5 OF 5 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 96305423 MEDLINE
DOCUMENT NUMBER: 96305423 PubMed ID: 8766570
TITLE: Accessory signaling by CD40 for T cell activation: induction of Th1 and Th2 cytokines and synergy with interleukin-12 for interferon-gamma production.
AUTHOR: Peng X; Kasran A; Warmerdam P A; **de Boer M**; Ceuppens J L
CORPORATE SOURCE: Department of Pathophysiology, Catholic University of Leuven, Belgium.
SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1996 Jul) 26 (7) 1621-7. Journal code: EN5; 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199609
ENTRY DATE: Entered STN: 19960924
Last Updated on STN: 19970203
Entered Medline: 19960916

AB The interaction of CD40 ligand (CD40L) on activated T cells with CD40 on B cells, monocytes and dendritic cells is essential for humoral immunity and for up-regulation of antigen-presenting cell (APC) functions, as a result of signaling through CD40. There are also some indications that after interaction with CD40, CD40L can directly signal T cells. In this study we demonstrate that upon stimulation of human peripheral blood T cells through the T cell receptor (TCR)/CD3 complex, CD40/CD40L interaction strongly enhances the production of Th1 cytokines such as interleukin (IL)-2 and interferon (IFN)-gamma and Th2 cytokines such as IL-4, IL-5 and IL-10 by a direct effect on T cells. Furthermore, CD40/CD40L interaction synergizes with **IL-12** in selectively enhancing IFN-gamma production by purified anti-CD3-stimulated T cells. These effects were observed at both the protein and the mRNA level. Both CD4+ and CD8+ T cells were able to produce IFN-gamma in the presence of helper signals from **IL-12** and CD40, although CD8+ T cells were less active. Since CD40/CD40L interaction also up-regulates **IL-12** production and B7 expression by APC, our results suggest that CD40/CD40L interaction is bidirectional, and promotes activation of both APC and T cells.

AU Peng X; Kasran A; Warmerdam P A; **de Boer M**; Ceuppens J L

AB . . . Th2 cytokines such as IL-4, IL-5 and IL-10 by a direct effect on T cells. Furthermore, CD40/CD40L interaction synergizes with **IL-12** in selectively enhancing IFN-gamma production by purified anti-CD3-stimulated T cells. These effects were observed at both the protein and the. . . mRNA level. Both CD4+ and CD8+ T cells were able to produce IFN-gamma in the presence of helper signals from **IL-12** and CD40, although CD8+ T cells were less active. Since CD40/CD40L interaction also up-regulates **IL-12** production and B7 expression by APC, our results suggest that CD40/CD40L interaction is bidirectional, and promotes activation of both APC. . .

=> s l5 and (IL-12 (5N) receptor) and monoclonal
L10 0 L5 AND (IL-12 (5N) RECEPTOR) AND MONOCLONAL

<------User Break------>
u
=> s 3H4 and IL-12?
TERM '12?' EXCEEDED TRUNCATION LIMITS - SEARCH ENDED
You have entered a truncated stem which occurs in too many terms.
Make the stem longer and try again. For example, if your original term was 'degr?' to search for variations and the abbreviation for 'degradation', you could replace it with the expression '(degrdn OR degrad?)'. If your search term was numeric, e.g., 'C>5', reduce the size of the range.

=> s 3H4 and (IL-12R or IL12 or (interleukin (1N) 12))
L12 1 3H4 AND (IL-12R OR IL12 OR (INTERLEUKIN (1N) 12))

=> dis l12 ibib

L12 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:350691 CAPLUS
DOCUMENT NUMBER: 130:351231
TITLE: Monoclonal antibody to the **interleukin-12** receptor .beta.2-chain
INVENTOR(S): De Boer, Mark; Den Hartog, Marcel Theodorus
PATENT ASSIGNEE(S): Tanox Pharma B.V., Neth.
SOURCE: PCT Int. Appl., 24 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9925737	A1	19990527	WO 1998-NL663	19981119

W: CA, JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: EP 1997-203607 19971119
REFERENCE COUNT: 7
REFERENCE(S): (1) BASF Aktiengesellschaft; WO 9841232 A 1998 CAPLUS
(2) F Hoffman La Roche AG; EP 0638644 A 1995 CAPLUS
(3) F Hoffman La Roche AG; EP 0759466 A 1997 CAPLUS
(4) Hoffman-La Roche Inc; US 5852176 A 1998 CAPLUS
(5) Hoffman-La Roche Inc; US 5853721 A 1998 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> end
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LOGOFF? (Y)/N/HOLD:y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	114.27	114.42
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CA SUBSCRIBER PRICE	-6.82	-6.82

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 NEWS  3  Feb 06  Engineering Information Encompass files have new names
 NEWS  4  Feb 16  TOXLINE no longer being updated
 NEWS  5  Apr 23  Search Derwent WPINDEX by chemical structure
 NEWS  6  Apr 23  PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
 NEWS  7  May 07  DGENE_Reload
 NEWS  8  Jun 20  Published patent applications (A1) are now in USPATFULL
 NEWS  9  JUL 13  New SDI alert frequency now available in Derwent's
                  DWPI and DPCI
 NEWS 10  Aug 23  In-process records and more frequent updates now in
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 NEWS 13  Sep 17  IMSworld Pharmaceutical Company Directory name change
                  to PHARMASEARCH
 NEWS 14  Oct 09  Korean abstracts now included in Derwent World Patents
                  Index
 NEWS 15  Oct 09  Number of Derwent World Patents Index updates increased
 NEWS 16  Oct 15  Calculated properties now in the REGISTRY/ZREGISTRY File
 NEWS 17  Oct 22  Over 1 million reactions added to CASREACT
 NEWS 18  Oct 22  DGENE GETSIM has been improved
 NEWS 19  Oct 29  AAASD no longer available
 NEWS 20  Nov 19  New Search Capabilities  USPATFULL and USPAT2
 NEWS 21  Nov 19  TOXCENTER(SM) - new toxicology file now available on STN
 NEWS 22  Nov 29  COPPERLIT now available on STN
 NEWS 23  Nov 29  DWPI revisions to NTIS and US Provisional Numbers
 NEWS 24  Nov 30  Files VETU and VETB to have open access
 NEWS 25  Dec 10  WPINDEX/WPIDS/WPIX New and Revised Manual Codes for 2002
 NEWS 26  Dec 10  DGENE BLAST Homology Search
 NEWS 27  Dec 17  WELDASEARCH now available on STN
 NEWS 28  Dec 17  STANDARDS now available on STN
 NEWS 29  Dec 17  New fields for DPCI
 NEWS 30  Dec 19  CAS Roles modified
 NEWS 31  Dec 19  1907-1946 data and page images added to CA and CAplus

 NEWS EXPRESS  August 15 CURRENT WINDOWS VERSION IS V6.0c,
               CURRENT MACINTOSH VERSION IS V6.0 (ENG) AND V6.0J (JP),
               AND CURRENT DISCOVER FILE IS DATED 07 AUGUST 2001
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L1           3 2-4E6

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PROCESSING COMPLETED FOR L1
L2             1 DUP REM L1 (2 DUPLICATES REMOVED)

=> dis l2 ibib abs kwic

L2   ANSWER 1 OF 1     MEDLINE                          DUPLICATE 1
ACCESSION NUMBER:    96206137     MEDLINE
DOCUMENT NUMBER:     96206137   PubMed ID: 8617302
TITLE:               Biological function and distribution of human
                     interleukin-12 receptor beta chain.
AUTHOR:              Wu C Y; Warrier R R; Carvajal D M; Chua A O; Minetti L J;
```

Chizzonite R; Mongini P K; A S; Gubler U; Presky D H; Gately M K

CORPORATE SOURCE: Department of Inflammation/Autoimmune Diseases, Hoffmann-La Roche Inc., Nutley, USA.

SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1996 Feb) 26 (2) 345-50. Journal code: EN5; 1273201. ISSN: 0014-2980.

PUB. COUNTRY: GERMANY: Germany, Federal Republic of

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199606

ENTRY DATE: Entered STN: 19960620
Last Updated on STN: 19960620
Entered Medline: 19960613

2.4E6

See Chua et al

5 536 657

AB We previously described the cloning of a cDNA encoding an interleukin-12 receptor (IL-12R) subunit, designated beta, that bound IL-12 with low affinity when expressed in COS cells. We now report that a pair of monoclonal antibodies (mAb), 2B10 and **2.4E6**, directed against different epitopes on the IL-12R beta chain, when used in combination, strongly inhibited IL-12-induced proliferation of activated T cells, IL-12-induced secretion of interferon-gamma by resting peripheral blood mononuclear cells (PBMC), and IL-12-mediated lymphokine-activated killer cell activation. The mAb had no effect on lymphoblast proliferation induced by IL-2, -4, or -7. Thus, the IL-12R beta chain appears to be an essential component of the functional IL-12R on both T and natural killer (NK) cells. We previously observed that high affinity IL-12R were expressed on activated T and NK cells, but not B cells. Studies using flow cytometry and reverse transcription-polymerase chain reaction analysis showed that IL-12R beta chain was expressed on several human T, NK, and (surprisingly) B cell lines, but not on non-lymphohematopoietic cell lines. The Kit225/K6 (T cell) and SKW6.4 (B cell) lines were found to express the greatest amounts of IL-12R beta chain (800-2500 sites/cell); however, Kit225/K6 but not SKW6.4 cells bound IL-12. Similar to SKW6.4 B cells, activated tonsillar B lymphocytes expressed IL-12R beta chain but, consistent with previous results, did not display detectable IL-12 binding. Likewise, up to 72% of resting PBMC from normal volunteer donors expressed IL-12R beta, but did not bind measurable amounts of IL-12. These results indicate that expression of IL-12R beta is essential, but not sufficient, for expression of functional IL-12R. We speculate that expression of functional, high-affinity IL-12R may require the presence of a second subunit that is more restricted in its expression than IL-12R beta.

AB . . . with low affinity when expressed in COS cells. We now report that a pair of monoclonal antibodies (mAb), 2B10 and **2.4E6**, directed against different epitopes on the IL-12R beta chain, when used in combination, strongly inhibited IL-12-induced proliferation of activated T. . .

=> end
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	5.93	6.08

STN INTERNATIONAL LOGOFF AT 14:09:15 ON 07 JAN 2002

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Search Results - Record(s) 1 through 3 of 3 returned.

☑ 1. Document ID: US 6054487 A

L1: Entry 1 of 3 File: USPT Apr 25, 2000

US-PAT-NO: 6054487
DOCUMENT-IDENTIFIER: US 6054487 A

TITLE: Methods and compositions for modulating responsiveness to corticosteroids

DATE-ISSUED: April 25, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Sekut; Les	Westborough	MA		
Carter; Adam	Newburyport	MA		
Ghayur; Tariq	Grafton	MA		
Banerjee; Subhashis	Shrewsbury	MA		
Tracey; Daniel E.	Harvard	MA		

US-CL-CURRENT: 514/604; 514/602, 514/603

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☑ 2. Document ID: US 5831007 A

L1: Entry 2 of 3 File: USPT Nov 3, 1998

US-PAT-NO: 5831007
DOCUMENT-IDENTIFIER: US 5831007 A

TITLE: Human receptor for interleukin-12

DATE-ISSUED: November 3, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Chua; Anne On	Wayne	NJ		
Gubler; Ulrich Andreas	Glen Ridge	NJ		

US-CL-CURRENT: 530/350; 530/351

Full | Title | Citation | Front | Review | Classification | Date | Reference KWIC | Draw Desc | Image

☑ 3. Document ID: US 5536657 A

L1: Entry 3 of 3 File: USPT Jul 16, 1996

US-PAT-NO: 5536657
DOCUMENT-IDENTIFIER: US 5536657 A

TITLE: Recombinant DNA encoding human receptor for interleukin-12

DATE-ISSUED: July 16, 1996

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Chua; Anne O.	Wayne	NJ		
Gubler; Ulrich A.	Glen Ridge	NJ		

US-CL-CURRENT: 435/252.3; 435/320.1, 435/69.1, 435/69.52, 536/23.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWIC | Draw. Desc | Image

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Terms	Documents
((IL12 or (IL adj 12) or (Interlekin12) or (Interleukin adj 12)) adj (R or receptor)) near monoclonal	3

Display 10 Documents, starting with Document: 3

Display Format: CIT Change Format

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Search Results - Record(s) 1 through 3 of 3 returned.

1. Document ID: US 6054487 A

L1: Entry 1 of 3 File: USPT Apr 25, 2000

DOCUMENT-IDENTIFIER: US 6054487 A
TITLE: Methods and compositions for modulating responsiveness to corticosteroids

BSPR:
The invention also provides pharmaceutical compositions for modulating responsiveness to corticosteroids in a subject. In one embodiment, a composition of the invention comprises an agent which antagonizes a factor that regulates production of IFN-.gamma. in the subject, a corticosteroid and a pharmaceutically acceptable carrier. In another embodiment, a composition of the invention comprises an IGIF antagonist (such as inhibitor of a caspase family protease, preferably an ICE inhibitor, or an anti-IGIF or anti-IGIF receptor monoclonal antibody), a corticosteroid and a pharmaceutically acceptable carrier. In yet another embodiment, a composition of the invention comprises an IL-12 antagonist (e.g., an anti-IL-12 or anti-IL-12 receptor monoclonal antibody, a phosphodiesterase IV inhibitor, a beta-2 agonist, a STAT4 inhibitor), a corticosteroid and a pharmaceutically acceptable carrier. The pharmaceutical compositions of the invention can be formulated for administration via a preferred route of administration for achieving a desired therapeutic effect. In one preferred embodiment, the pharmaceutical composition is formulated for topical administration. In another preferred embodiment, the pharmaceutical composition is formulated for administration by inhalation. Other preferred routes of administration include oral and intravenous administration.

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWIC | Draw Desc | Image

2. Document ID: US 5831007 A

L1: Entry 2 of 3 File: USPT Nov 3, 1998

DOCUMENT-IDENTIFIER: US 5831007 A
TITLE: Human receptor for interleukin-12

DEPR:
The murine anti human IL-12 receptor monoclonal antibody 2-4E6 used herein was generated as described herein below in Examples 1 to 16 and was purified from ascites fluids by affinity chromatography on protein G-agarose according to the manufacturer's instructions (Genex). The proteins were labeled with 1-125 by a modification of the Iodogen method as described (Pierce Chemical Co., Rockford, Ill.). Radiospecific activities of 5000-7000 cpm/fmole for IL-12 and 1500-2500 cpm/fmole for the 2-4E6 antibody were typically obtained.

DEPR:
The murine anti human IL-12 receptor monoclonal antibody 2-4E6 was prepared, characterized, and generated as set forth in U.S. patent application Ser. No. 08/094,649, filed Jul. 19, 1993, which has been refiled as a continuation-in-part application Ser. No. 08/248,532, filed May 31, 1994, now abandoned the contents of both applications being expressly incorporated by reference herein and is as follows:

Full | Title | Citation | Front | Review | Classification | Date | Reference | Claims | KWMC | Draw. Desc | Image

3. Document ID: US 5536657 A

L1: Entry 3 of 3 File: USPT Jul 16, 1996

DOCUMENT-IDENTIFIER: US 5536657 A
TITLE: Recombinant DNA encoding human receptor for interleukin-12

DEPR:
The murine anti human IL-12 receptor monoclonal antibody 2-4E6 used herein was generated as described herein below in Examples 1 to 16 and was purified from ascites fluids by affinity chromatography on protein G-agarose according to the manufacturer's instructions (Genex). The proteins were labeled with 1-125 by a modification of the Iodogen method as described (Pierce Chemical Co., Rockford, IL). Radiospecific activities of 5000-7000 cpm/fmole for IL-12 and 1500-2500 cpm/fmole for the 2-4E6 antibody were typically obtained.

DEPR:
The murine anti. human IL-12 receptor monoclonal antibody 2-4E6 was prepared, characterized, and generated as set forth in U.S. patent application Ser. No. 08/094,649, filed Jul. 19, 1993, now abandoned, which has been refiled as a continuation-in-part application Ser. No. 08/248,531, filed , May 31, 1994, the contents of both applications being expressly incorporated by reference herein and is as follows:

Full | Title | Citation | Front | Review | Classification | Date | Reference | KWMC | Draw. Desc | Image

Generate Collection

Terms	Documents
((IL12 or (IL adj 12) or (Interlekin12) or (Interleukin adj 12)) adj (R or receptor)) near monoclonal	3

Display 10 Documents, starting with Document: 3

Display Format: KWIC Change Format